Pneumocephalus in Child Following Bilateral Otomastoiditis and Nasal Septum Infection

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ABSTRACT
Pneumocephalus is collection of gas or air within the cranial cavity, commonly associated with trauma, cranial surgery, air embolism, open meningomyelocoele; and rarely as a result of central nervous system infections. Asymptomatic pneumocephalus usually recovers spontaneously within few days. Untreated pneumocephalus can progress to tension pneumocephalus, manifesting as severe headache, dizziness, cranial nerve palsy, mental changes, seizure and disorientation. Herein, we report a rare case of pneumocephalus in a 9-month infant with subdural effusion following infection of nasal septum and otomastoiditis. There was no sign of meningitis but CT head showed communication of intracranial dura mater across widened foramen caecum with pre-nasal space, and bilateral otomastoiditis with erosion of anterior and lateral wall of right mastoid bone. The patient was treated successfully and discharged without sequelae.


INTRODUCTION
Pneumocephalus, the presence of air within the cranial cavity, is most commonly caused by trauma, pyogenic meningitis, brain abscess, open myelomeningocele, para-nasal sinusitis, facial tumors that eroded through skull, post-radiation necrosis of skull appendages or is iatrogenic - following neurosurgery, positive pressure ventilation, facial procedures or pneumo-encephalogram.1-5 Pneumocephalus has been reported in adults associated with meningitis caused by various aerobic and anaerobic organisms including Streptococcus pneumoniae, Staphylococcus aureus, Enterobacter cloacae, Klebsiella aerogenes, enterococcus, Candida albicans, Clostridium perfringens, and Bacteroides fragilis in the absence of other risk factors for pneumocephalus.6-10 It is rare in infants as described hereby.

CASE REPORT
A 9-month male infant was admitted to our hospital with complaints of fever, loose motions and bilateral ear discharge for 15 days. He had a boil on the tip of his nose, 15 days back. It burst spontaneously and led to erosion of nasal septum. Prior to admission to the hospital, parents were seeking treatment for their child on outdoor basis without any improvement. There was no history of facial trauma, seizures, recurrent infections or a history suggestive of an immune-compromised state. There was no family history suggestive of tuberculosis.

Infant was irritable, febrile (101˚F) and had difficulty in breathing (RR=54 breaths/minute) at the time of admission. The infant had right-sided lower motor neuron type facial palsy with no other neurological and cardiovascular abnormality. Blood investigations revealed leucocytosis (TLC=38380/mm³) with predominantly neutrophils (polymorphs=88%, lymphocytes=9%). Cerebrospinal fluid examination showed 4 cells/mm³ (all lymphocytes), proteins = 24 mg%, sugar = 78 mg% and negative Zach-Nelson and Gram's staining. Cerebrospinal fluid culture for aerobic and anaerobic organism was sterile, HIV and VDRL tests were non-reactive. Blood culture (bactec alert) was sterile. Ear swab culture revealed growth of Pseudomonas sensitive to amikacin, piperclillin and tazobactum. Nasal swab, ear swab and CSF were sterile for fungal culture. His serum IgM (40 mg/dl), IgG (475 mg/dl), and IgA (48 mg/dl) were within normal limit.

His brain stem evoked response audiometry examination revealed bilateral hearing impairment. Computed tomography (CT) of head showed subdural effusion with pneumocephalus, bilateral frontal lobe compression, peaking and separation of the frontal lobes from frontal bone “Mount Fuji” sign (Figure 1). Sagittal reformations in bone algorithms show communication of intracranial dura mater across widened foramen caecum with pre-nasal space. Coronal reformations in bone algorithms show widened foramen caecum, ethmoid and maxillary sinus fluid, discontinuity of cartilaginous nasal septum. Nasal bones, ethmoid septae and bony nasal septum were intact. Temporal bone showed bilateral otomastoiditis with erosion of anterior and lateral wall of right mastoid bone, ossicles appear intact bilaterally (Figure 2).
Patient was managed conservatively. He was kept under observation, treated for bilateral otitis media, mastoiditis and pustule at the tip of nose with Inj. Amikacin and pipercillin and tazobactum. Child was improved and discharged. Computed tomography head done 2 weeks later showed complete resolution of pneumocephalus and reduction in the size of subdural effusions.

**DISCUSSION**

Subarachnoid or subdural air accumulation is the most common form of pneumocephalus and intracerebral pneumocephalus can occur *albeit* rarely.

Andrews and Canalis reported trauma as a cause of pneumocephalus in 36%, otitis media in 31%, otitic surgery in 31%, and congenital defects in 2%. Pneumocephalus occurs either following traumatic fracture (the paranasal sinuses) or intracranial anaerobic and aerobic infections. In cases with intracranial infection, the organisms may produce gas by either putrefaction of the intracellular protein derived from autolysis or by decomposition of glucose. Ischemia may also be a contributory factor in which any gas produced may not be absorbed. These factors may act singly or in combination, resulting in pneumocephalus.

Frontal pneumocephalus is the most common type of pneumocephalus. About 75 - 90% of pneumocephalus are post-traumatic and the incidence of pneumocephalus secondary to head injury ranges from 0.5% to 13.2%. Facial fractures (0.5% - 13.2%), involving the ethmoidal or frontal sinuses, are associated with dural tear, osteo-meningeal fistula, CSF rhinorrhea and pneumocephalus. It develops either as acute or subacute complications of head trauma, or as late complications during the recovery phase of head injury.

Rarely pneumocephalus may be seen as a complication of positive airway pressure ventilation. Positive pressure in the upper airway can force air into the skull through any openings of the facial bone. The other mechanism of air penetration is a ball-valve effect like sudden increase of nasopharyngeal pressure, during coughing and sneezing, forcing air into the cranial cavity which subsequently remained trapped.

The asymptomatic pneumocephalus usually recovers spontaneously within few days. Untreated pneumocephalus can progress to tension pneumocephalus, manifesting as severe headache, dizziness, cranial nerve palsy, mental changes and even seizure and disorientation. Therefore, it requires emergency surgical intervention - subdural drainage of air to decrease intracranial pressure – immediately after confirming the diagnosis either with X-rays skull or CT head. Its further management includes: strict bed rest in the semi-fowler position, analgesia and antibiotics, high FiO2 oxygen to reduce blood and brain tissue nitrogen concentration, and by increasing the nitrogen concentration gradient that promotes pneumocephalus gas absorption.

Further increase of intracranial pressure can be prevented by avoiding the Valsalva manoeuvre, coughing, sneezing, nose blowing, and excessive physical activities.

As this patient was 9 months of age with open anterior fontanelle, so tension pneumocephalus did not develop. Though he presented with right-sided lower motor neuron type facial palsy but had no other neurological abnormalities or signs of tension pneumocephalus; and was, therefore, managed conservatively. The outcome was an uneventful recovery.

**REFERENCES**


